

THE ALCOHOLIC PSYCHOSES.  
CHRONIC ALCOHOLIC DELIRIUM (KORSAKOFF'S  
PSYCHOSIS).

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## THE ALCOHOLIC PSYCHOSES. CHRONIC ALCOHOLIC DELIRIUM (KORSAKOFF'S PSYCHOSIS).

*A Clinical Lecture.*

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I have already pointed out the immense rôle that the various types of intoxications play in the development of mental disturbances. We have seen that the toxic, infectious, and exhaustion psychoses have many signs in common, and that it may, for instance, be impossible to distinguish the mental disturbance due to the toxine of the influenza bacillus, from that due to the typhoid organism, i. e., seen purely from the mental side.

This same truth is to be seen in a much less marked form in the consideration of the toxæmias of a genesis, chemically speaking, much more definite. For in the specialized type of mental disturbance, Korsakoff's psychosis—or polyneuritic psychosis—of which we shall speak, it is well known that the symptomatology will show a fairly uniform picture, notwithstanding a very marked variation in ætiology—alcohols, certain metals, lead, arsenic, diabetes, tuberculosis, carcinoma, puerperal infection, etc.<sup>1</sup>

We have just opened the discussion on alcohol in its relations to mental disturbance, and I have

<sup>1</sup>Compare Dupré. *Traité de pathologie mentale*, pp. 1131-1140, where a full list of ætiological factors, some twenty-six or twenty-seven, are listed.

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given you a picture of the effect of alcohol in its power to dissociate the mechanisms of both mental and neural integration. We have gone hastily over the subject of acute drunkenness, which from a medical point of view is a very definite psychosis. Whether it should be regarded as an insanity from the standpoint of legal requirements is a knotty problem. We have discussed that type of subacute or chronic poisoning known as delirium tremens; noted the extreme variability in its various aspects, and I have tried to show that in the analysis of this type of alcoholic delirium we may find many practical suggestions as to how to study other mental disturbances. The relation of acute hallucinosis to delirium tremens and to other forms of mental disturbance has been pointed out. You have been warned not to confound this type of an alcoholic psychosis with other more sinister mental disturbances.

There remains for discussion but two other fairly well crystallized groups in this alcoholic medley—those forms which show a tendency to chronic delusional development—the so called alcoholic paranoias, and the mental disturbance which is associated with a more or less marked degree of polyneuritis, the so called polyneuritic psychosis, or Korsakoff's psychosis. We shall take up the latter this afternoon.

*History.*—Alcohol has been the boon companion of man ever since we have any records. Acute and chronic drunkenness have probably always existed, and it is inconceivable that multiple neuritis with marked mental symptoms did not occur in ancient Greece and Rome; yet we cannot credit the Greeks this time with the knowledge of a polyneuritic psychosis. Magnus Huss has recorded that mental symptoms accompany neuritis as early as 1849.

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The term Korsakoff's psychosis is more or less popular at the present time: Jolly, professor at Berlin, having used the term first in 1897. Up to the present time no one has shown that Korsakoff's description is not the first fairly complete and accurate account that we have. It is true that Jackson, in America, as early as 1822, in describing alcoholic multiple neuritis, says that in these cases "the mind is weakened, but that it is free from delirium ordinarily," from which by inference we learn that Jackson recognized the occasional presence of delirium.

Dr. M. Allen Starr, in his Middleton Goldsmith lectures in 1887, on Multiple Neuritis and its Relation to Certain Peripheral Neuroses, *Medical News*, February 5, 1887, p. 141, makes the following remarks concerning the mental features in certain cases of multiple neuritis:

One feature of alcoholic paralysis remains to be noted, viz., the cerebral symptoms. These are hardly ever wanting. There is, first, the excitement rising to the degree of active delirium, with illusions and hallucinations of the various senses; there is the insomnia which so soon exhausts the patient if it is not remedied; there is the loss of memory, especially of recent occurrences; and the lack of power of attention or concentration which prevents intelligent conversation. The indifference to bodily wants may be so great as to lead to uncleanliness, and since paralysis of the sphincter is the rare exception, incontinence is usually to be ascribed to the mental state. It is impossible to get any reliable history of their illness from these patients. Their statements are unintelligible or unreliable; and here it may be well to notice a symptom first described by Strümpell.<sup>2</sup> These patients will relate occurrences as having happened recently with such elaboration of detail, when, as a matter of fact, the story is entirely a product of the imagination. Thus, one patient of my own, who had been confined to bed for many days, told me one afternoon that she had been out to see an eminent gynaecologist during the morning, had gone to his office and waited

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<sup>2</sup>*Archiv für Psychiatrie*, xiv, p. 339, 1883.

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for him several hours; had seen other patients there, and finally had been told by the doctor's brother that he would not return in time to see her, so she had come home again. And this was all related in apparent good faith, so that I have no doubt that she believed that what she said had occurred. With the possibilities of such delusions in view, it is evident that the statements of these patients cannot be accepted regarding anything, especially as to their own history. One patient who was admitted to Bellevue, during my service there, told me a different story of her case every day for a week, and it was only by interviewing her friends that the correct account was obtained.

In Starr's account it cannot be said that he grasped the causes for the falsifications of memory. He calls these statements delusions.

Names, after all, are but symbols standing for things, and the only requirement that science demands is that when a symbol is used, the user should have a clear and thorough idea of what is meant. It is for this reason that I shall quote to you at some length fragments from descriptions given by Korsakoff himself—then a privatdocent in the University of Moscow. I cannot give you his earliest descriptions, as they are published in Russian,<sup>3</sup> but in 1890 (*Archiv für Psychiatrie*, xxii, p. 669), under the title of Ueber eine besondere Form psychischer Störung combinirt mit multipler Neu-  
ritis, he first calls attention to his earlier paper, published in (*Vestnik Dushevnikl Polieznei*, iv, No. 2) 1887, in which he describes a peculiar form of mental disturbance occurring in cases of multiple neuritis of nonalcoholic origin. This psychic disturbance is sometimes characterized by a very definite irritable weakness in the psychic sphere, at times in the form of confusion with marked characteristic mistakes with reference to place, time, and situation,

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<sup>3</sup>S. Korsakoff. *Die Störung der psychischen Tätigkeit bei Alcohol-neuritis und ihre Beziehung zur Störung auf dem psychischen Gebiete bei Polyneuritiden nicht alcoholschen Ursprungs*, as the title appears translated into German by the author himself.

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and at times as a nearly pure form of acute amnesia in which memories of very recent events are profoundly modified, while events which took place in the past remain comparatively intact. Such a form of psychic disturbance, he writes, is highly characteristic of multiple neuritis of alcoholic origin. It does not, however, belong to alcoholic neuritis exclusively, but occurs in forms of neuritis due to other toxic agents, chief of which he held were toxæmias of gastrointestinal origin.

Korsakoff was aware of the fact that in literature there had been cases of multiple neuritis reported, in which psychic symptoms had been noted, but such mental abnormalities had not been carefully analyzed. In the article which I show you he reports two of his earlier cases showing the characteristic mental symptoms, in whom the neuritis was not of alcoholic origin. He then proposes the name cerebropathia psychica toxæmica--then follows the description of six cases of alcoholic multiple neuritis with the characteristic symptom picture.

These are the six cases which I have had the opportunity to observe in the past two years. They all have much in common; in the first place the psychic alterations bear a close resemblance. It is true that these vary, in one case being more marked, in another milder—but in all the most prominent symptom was a more or less deep disturbance of association of ideas and of the memories. In the severer cases there developed a complete loss of relation between the single ideas, while in the milder cases the ideas simply were mixed, the memories were tossed about, and forgetfulness was very striking. In all of these cases it was specially characteristic that there was a marked confusion with reference to different occurrences, which often had no foundation in fact, which the patient would narrate in the most natural manner. In one case there was added to this condition a state of emotionalism, of anxiety, with accompanying hallucinations and illusions.

The presence of a neuritis was a second characteristic present in all cases, although varying considerably in its in-

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tensity. All cases showed weakness in gait, the rectus femoris being involved.

Speech disturbances were noted by him, and in one case nystagmus. Summing up (p. 700) Korsakoff gives the following very terse description, which naturally has been somewhat enlarged at the present time:

The fundamental symptoms of the disease are, as a rule, the following: A high grade of irritable weakness of the psychic spheres, then a more or less deep disturbance of association of ideas, and finally a clouding of memories. The weakest expression of the involvement of the psychic spheres in this disease seems to be an irritable weakness which shows itself in sleeplessness, in easy tire of the brain, which gives rise to fear and anxiety. Toward evening especially, the patients are easily excited, fear something, wait for something, literally are discontented with everything. To this, not uncommonly, may be added inability to concentrate the attention and the impossibility of ridding themselves of certain ideas, by reason of which compulsory ideas arise which are often of exciting or fearful nature.

Associated with these ideas arise many strange and impossible wishes. If the psychic disturbances go deeper the impossibility of the correct effort of thought becomes apparent, the attention is not any longer in a position to lead to the association of ideas, these ideas therefore become mixed, inconsequent, and incorrect in consciousness. Such a condition may develop acutely even in the beginning of the disease, or simultaneously with the initial symptoms of the multiple neuritis, occasionally before. In the majority of cases there arises in the beginning a condition of strong emotional disturbance, chiefly in the form of fear (panophobia) accompanied by distinct delirium, hallucinations, and emotional conduct. This irritable condition, as a rule, does not last very long, but either passes over into recovery or into a chronic state. This chronic form bears the character of a stuporous dementia, or an apathetic confusion.

The stuporous dementia shows itself in a deep disturbance of judgment, with associated delirious ideas, illusions, and hallucinations, often with short attacks of fury. In some cases the dementia reaches a very high grade, the patients become weak minded, uncleanly. During this period the symptoms of multiple neuritis such as an irregular, weak gait, disturbances of the patellar reflex, tremor of the

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extremities, sometimes due to the involvement of the cranial nerves, may all be present, so that one may confound the disease with paresis, and be surprised to find the patient get well. For the most part, however, such cases of neuritic pseudoparalysis are comparatively few.

Cases with simple stupor, or with temporary excitement where the patients either get well in from five to nine months, or remain unchanged are the more common.

In other cases the chronic form of this disorder shows the character of an apathetic confusion, occasionally as an end stage there arises an acute hallucinatory fury with confusion, in other cases it develops gradually without any going on to such a furious stage. It is characterized by confusion of ideas, disorientation with reference to time and place, numerous mistakes and weaknesses in memory. The patients often do not know where they are, even when they are in their own rooms, confound the persons about them, call them by the names of those long dead, accredit to themselves actions which they have never done. These patients mix in a remarkable manner ideas of real facts with old memories to form very peculiar combinations of thought; the memory is as a rule deeply disturbed, the patients often forgetting everything of what goes on around them. As a rule such patients are comparatively quiet, apathetic, emotional states do not arise, at times there is a mild tendency to laugh or cry, and many patients who are quiet in the day-time are excitable at night, talk constantly, cry aloud, are quarrelsome, want to get up and go about.

This form of confusion is most common, it appears in more marked, or less marked degrees, sometimes passing away with comparative rapidity, again persisting for a long time. For the most part it increases gradually and becomes so marked that the patient not only confounds acquaintances with strangers, but loses entirely the significance of objects, words, and signs.

I have already said that in this form there is nearly always a clouding of memory, of a greater or lesser intensity, but there do occur forms of psychoses in neuritis in which the memory is disturbed, but there is a relative clarity of consciousness and of judgment. In these cases it is highly striking how the same patient, who is able to grasp clearly everything about him, who is able to follow serious conversation, yet whose memory is so distinctly involved that he literally forgets everything that has been said, and this forgetfulness reaches such a grade that the patient, five minutes after his lunch, does not remember

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that he has eaten it, he forgets with whom he has been, and what he has said. Amnesiae of this type (amnesia acuta) limit themselves mostly in that occurrences which have taken place since the beginning of the disease, or shortly before, are forgotten, whereas that which has occurred in times long past often remains clear in the memory. The more severe the case, as a rule, the deeper is the amnesia. In severe cases the memory of old events may also be bad, and in the mild cases recent events may not be entirely forgotten, but there is above all a forgetfulness, an inaccurate memory of time, etc., apparent.

The symptoms of the multiple neuritis, and the appearance in the peripheral nervous system are not always developed in the same way in this disorder. In some cases they preponderate so that the psychic alterations, consisting for the most part only in an irritable weakness, constitute the background. Thus in some there arises a very severe form of multiple neuritis, which runs the course of an acute ascending paralysis, in others there is a long drawn out severe form of paralysis, hyperalgesia contributes and muscle atrophies. In other forms the peripheral and psychic symptoms show with equal force, and finally there are other forms in which the physical symptoms of neuritis may be present in mild degree, and may be entirely overlooked. This stage may be limited solely to the occurrence of paræsthesiæ in the extremities (feeling of coldness, feeling of binding about the feet or hands, tickling as of crawling of ants) with mild disturbances of coordination, and ready fatigability in walking associated with alterations of the patellar reflexes, and mild pains in the extremities. Such cases should awaken our special interest since they come mostly to the attention of the practitioner.

In addition to the appearance of multiple neuritis, other symptoms appear which indicate disturbances of the nervous system; often they point to a local disease of the brain. Again the symptoms are those of a spinal cord disorder, then they seem to point to an independent disease of the muscles, a myositis which may be spread over many muscles. Nearly always there are signs which prompt one to state that the entire body is involved. General loss of flesh is noticed with a very distinct loss of power; the pulse is often very rapid and irregular and the urine is usually deep colored, in quantity, and shows a relatively high percentage of destructive products. Disturbances in the intestinal canal, obstinate vomiting, retained menses, and a slight increase of temperature, etc., are usually found.

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The course of the disease is also interesting and characteristic. For the most part it arises in consequence of a definite cause, whether that be exposure to cold, a previous acute disease, poisoning, or marked fatigue, or by a cachexia, which arises at the same time. Very frequently the disease begins with obstinate vomiting, following which the appearance of psychical disturbance in one or another form takes place. Frequently at the same time, often earlier, not seldom later the appearances on the part of the peripheral nervous system show themselves.

According to the onset whereby the disease is developed it may run an acute course, and reach its maximum in a short time. For the most part, however, it runs a subacute course, and less seldom a very slow chronic course occurs.

If the disease develops on the basis of an incurable cachexia it terminates with the death of the patient, if this is not the case, however, there follows after the acute period of the disease, a second period in which the condition improves, the psychic and physical symptoms recovering in parallel manner. There are certain cases in which the psychic symptoms clear up sooner than the physical ones, and vice versa, where symptoms of the psychic alteration still remain after the disappearance of the multiple neuritis.

The improvement is usually accompanied by a general increase in the strength, the interrupted functions again take their place, the body weight commences to improve, especially the functions of the nerves increase.

Very frequently there remains, after the cure, a very marked fatigability of the nervous system, especially in the psychic sphere, and a great tendency to a recurrence.

With this description in mind as an original from the founder of this disorder, let me turn your attention to a patient who was admitted to the City Hospital about ten days ago.

CASE I.—J. K., twenty-nine years of age, a traveling salesman, an active, intelligent, able man.

This patient, as you see him lying in bed, presents an interesting type of physiognomy, to which I would like to call your close attention. It is neither normal nor yet markedly abnormal. You see that it expresses a fair degree of alertness but is a trifle stupid save when he smiles, which, as you notice, he does not infrequently. This slightly stupid expression, when you come to analyze it, is depend-

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ent largely upon the fact that the skin is swollen, the lips are slightly swollen, the eyelids droop somewhat, the eyeballs are slightly injected, and the fine veins of the nose and cheeks are somewhat prominent. The color of the face is a trifle unhealthy, being a little less bright as far as the lips and conjunctivæ are concerned than they should be, as though there were some modification of the aeration of the blood, which is probably accounted for by the somewhat quick and altered breathing.

In response to a question as to his name he tells you, with a laugh, that it is somewhat silly, that it is J. K.; that he is twenty-nine years of age, that he has been a traveling salesman selling shoes, and on asking him where he is, you will notice that he looks around, and says that he guesses he is here, and on wanting to know where "here" is, he tells us that "it looks like a hospital," and in response to the question "How long have you been here?" he tells us that he arrived this morning (whereas as a matter of fact he has been here nearly ten days). You may have noted that as I gave him my hand to shake, as we approached the bed, he lay motionless and nodded, but beyond some ineffectual movements of the arms was unable to take my hand. We shall examine into this a little later. On asking him what is the matter with him, he tells us that he feels all right; "Why do you not get up and walk?" you will note that his response is that he was up all the morning, out on the roof, and that the day before he had been walking all day, and he did not feel like getting up just now.

As he talks, note that his speech is somewhat halting, that the muscles of his face show slight tremors, and that his tongue, as he shows it to us, is coated and tremulous. He was partially asleep, you will recall, as we approached the bed, and should you turn away, you will find that he will become somnolent again, or even seem stuporous.

Testing his sense of smell, his sight, his hearing, his taste, his feeling to pin prick, etc., you will note that he answers more or less correctly. You will note that when I first approached him, some four or five minutes ago, I introduced myself, and introduced you all as members of a class of young physicians interested in the study of mental disorders, and I now ask him what is my name, and you will notice that he is unable to reproduce it, and I tell it to him again at this time, and will ask you to remind me to ask him the same question again when we have finished our investigations. I will also tell him at this time the "polar bear" story from your *Outlines of Psychiatry* with which

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you are familiar, and will ask him to try and tell it to us again at the close of the clinic, impressing on him the necessity for his holding it in his memory.

Further you will notice that as I ask him again where he is he tells us that he is in the store. "What store?" "Macy's"; and should we test him still further you would find that there is a very distinct loss of orientation, which as we have seen already involves time, since he has not been out of bed for ten days, and yet he tells us that he only arrived to-day; and so far as orientation in place is concerned his answers are still more confused and contradictory.

Let us see what he did yesterday when he said he was out walking all day. In response to my suggestion, "You remember that we sat on the wall and were fishing," he says "O yes! and those were pretty good fish that we caught." And in response to my further suggestion that it was a fine haul, he says, "Yes, more than three men could carry home." and I then am able to lead him to the story of a horseback ride that he took this morning with me, and with one of you whom I point out and whom he remembers perfectly as having been with him.

I call particular attention to this type of suggestion confabulation in this disorder which we have before us. It is very typical. As he still further rambles on, you will notice that he gives a very highly colored picture of his activities for the past week, he tells us of deeds that he has performed, which, in spite of the fact that he has been helpless in bed, paralyzed in his hands and feet, do not seem to him to be at all incongruous.

You will notice that throughout all this conversation, my questions and his answers, that he is fairly alert, that his attention is fairly easily gained, but that he very rapidly wanders off into other fields which have nothing to do with the subject of the conversation, but that he can be very readily brought back again, showing a fairly light and easy grade of fixation of attention which, however, you can see is very superficial. You will notice, furthermore, that his grasp on things is very slight; that whereas he seems to understand what we are talking about it is very evident that, beyond the merest superficial hold on the subject, it does not penetrate very deeply into his thought processes. I have tested his book memory, his ability to multiply and subtract, to write the names of some of the Presidents of the United States, places in New York city with which he is familiar, and find out that his memory of things

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which have taken place in the past is fairly good, but that, for at least a period of six months, his memories are very poor, and are mostly supplanted by the confabulations of which we have already had ample illustration.

We did not notice the presence of any hallucinations, although when the patient at first came into the hospital at night he cried aloud, was uneasy, and gave evidence of having acute hallucinations of sight and of hearing. He was then in a mild state of delirium quite comparable to the delirium of delirium tremens.

Asking him if he is a heavy drinker, of course he says that he "drinks a little beer, and a little whiskey, well, once in a while," and he finally is made to confess that perhaps he sometimes drinks a little more than he ought, but we learn from his wife that he has been a hard drinker for the past six months and more, and, for days at a time has been drunk.

I next call your attention to his arms and legs. You notice that he is able to extend his wrists with considerable difficulty, that the whole forearm is weak; he has some power to grasp the hand, but you will observe that it is very weak. Examination of the feet shows that there is a mild degree of foot drop, that extension is almost gone, and as we get him up to his feet to walk, he is hardly able to stand, walks with support, with his feet far apart, and the exertion is almost too much for him. His knee jerks, ankle jerks, wrist jerks, and elbow jerks are all gone.

As I press upon the ulnar nerve, you notice that he winces, although my pressure is very slight. Pressure along the sciatic provokes the same painful sensation, and the entire body shows marked tenderness on pressure of the skin and muscles, especially over the nerve trunks. I call your particular attention to his pupils, which you will perceive are unequal, and do not react to light, although they do to accommodation. In other words, this patient shows a fairly typical Argyll Robertson pupil. Let me add that upon his admission, ten days ago, there was almost complete paralysis of accommodation, and that the convergence reaction was very slow. I call your particular attention to the presence of this Argyll Robertson pupil, since you will find it recorded in reliable textbooks that this condition is not found in the disease which we have before us.

A brief recapitulation of this case shows us a chronic drinker who has frequently been drunk and

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who came into the hospital unable to walk, with the signs of a multiple neuritis and an active delirium associated with hallucinations of sight and of hearing. That after he had lain in a semistuporous condition for a few days, his delirium abated, but instead of making a complete recovery, as many cases of delirium tremens do, we find him passing over into a chronic phase, in which he has some delirium at nights, although otherwise he is quiet and clear. His mood is more or less happy, and his general conduct is all that could be desired. He shows no marked alteration of personality and understands everything that is going on about him.

A physical examination shows unmistakable signs of polyneuritis, severe in grade—not causing complete paralysis of his arms or legs, yet involving the reflex mechanism of the eye.

Mentally he shows some very striking changes.

(1) In the first place there is a definite disturbance of his power of attention. He was not well oriented as to his surroundings, as to time, as to place, although this has improved somewhat. Now, at times, he seems to be correctly placed, and in half an hour later he may be distinctly lost. His sense of time is particularly involved, and he cannot tell within days how long he has been here.

(2) He further shows marked memory defects. A retrograde amnesia which stretches back several months. He has almost no ideas of what he has been doing for some time past.

(3) To make up for these obvious memory defects we noted that the patient attempted to fill up the gaps by telling us of things that he had done, which were manifestly impossible. This confabulation is promptly negatived by another and then still another, and the patient by suggestion may be led to say almost anything. You may recall how easily

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one may suggest hallucinations to patients suffering from delirium tremens. This patient thus shows us two definite types of confabulation, the so called *opportune confabulation*, i. e., an attempt to fill up the gaps in his memory, and the *suggestion confabulation* already spoken of. Sometimes the patients will show a rich phantasy confabulation, built up largely on a basis of a mild hallucinosis. It is almost a dreamy delirium, similar in type to that which Regis assures us Hercules suffered in Euripides's tragedy of the *Mad Hercules*. In some instances the confabulation approaches very closely the delusions of grandeur of the paretic. Yet alongside of this active confabulation the patient interrogated with reference to old events is usually pretty clear and orderly. This is not the invariable rule, however, since much depends on how widespread the cortex may be involved in the neuritic process.

Before passing to the history of another case, let me say a few words concerning the general pathology of Korsakoff's psychosis.

*Pathology.*

The lesions which have been described in chronic alcoholic delirium, or Korsakoff's psychosis, do not differ in any essential respect from those that we know of when studying multiple neuritis; the varying symptom picture depends exclusively upon the distribution of the lesions. Furthermore, it should not be forgotten that, not only is the nervous system very extensively implicated, but that other organs of the body are also involved, so that one has to consider, for the graver cases at least, that Korsakoff's psychosis is a combined result of localized neurological lesions as well as of a general toxæmia. A complete analysis of the pathological findings in

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the different cases would be beyond the purposes of this talk. I can only remind you of what I have said with reference to the distribution of the lesions in general paresis; it emphasizes the diversity in the clinical picture and its correlations in pathological involvement.

You will best understand the pathology of this intoxication by assuming the same type of degenerative changes in the neurons which are seen peripherally to be extended into the central nervous system. Thus the changes in the functions of the spinal nerves are to be explained largely by reason of peripheral involvement, and the changes in the psychical functions are due to more or less extensive intraneuronal disease. I shall again call to your mind the analogy which I outlined in discussing the subject of alcoholism, that alcohol is the great dissociator of function, and in the profound alteration in the psychosis in question we find that organic changes make permanent, to a greater or less extent, these dissociations which functionally are seen in the simpler types of ordinary drunkenness. It can thus be seen that Bonhoeffer, in assuming that chronic alcoholic delirium, which is his name for Korsakoff's psychosis, is practically nothing more than a prolonged and chronic delirium tremens, has not only the weight of evidence of symptomatology, but also the findings of pathological anatomy to support him.

It has only been in recent years that the degenerative changes in the brain stem, basal ganglia, and pallium itself have been subjected to critical analysis, more particularly by the methods of Marchi, Herxheimer, and Nissl. The initial changes are those of any type of acute toxic delirium; they need not detain us. The Marchi method utilized in the early stages of the disease, as

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Siefert has done in a case dying six days after the onset of the affection through a bronchial pneumonia, shows very widespread degeneration in the fibres of the pallium, the intracortical fibre network, and also the tangential fibres show signs of nutritive degeneration. In Siefert's case, in particular, the fibres of the central lobes bore the brunt of the degenerations.

Heilbronner has also found the same type of degenerative changes in the cortex, particularly in the central lobes, but he has also reported cases of two weeks' duration, and of six weeks' duration without any marked signs of degeneration. This latter finding is not difficult of interpretation, when we bear in mind how great a functional loss may be present without any necessarily permanent organic basis. In the various brains that have been examined an extraordinary amount of variability in the locations of the lesions has been observed, but in all cases one expects to find distinct changes in the fibres of the associated mechanisms, thus accounting for the clinical pictures. On the groundwork of the precise localization of such lesions one is unable on pathological foundation oftentimes to define between a Korsakoff syndrome of senility or of alcoholism. The bloodvessels usually show very characteristic changes. These are usually very widely dilated, and there is a tendency to an increase in the number of small bloodvessels, somewhat similar to that which has been observed in the new vessel proliferation of paresis, rarely, however, reaching such a severe grade. Hyaline degeneration of the walls is extremely common, whereas many of the vessel walls are thickened and arteriosclerotic. As in delirium tremens, so in chronic alcoholic delirium, there is a tendency to minute haemorrhagic extravasations. These are found not only in the cortex, but also in

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the basal ganglia and nerve nuclei, and even in the peripheral nerves. Such extravasations are usually localized along the course of the bloodvessels, and not infrequently give rise to large degenerative masses, thus accounting for the localizing symptoms so frequently observed in Korsakoff's psychosis and in polioencephalitis superior (Wernicke). In this latter disorder it is only necessary that the extravasations should occur in the neighborhood of the nuclei of the eye muscles.

Miliary haemorrhages are the rule, but occasionally they may be macroscopic in size. Thus in a case of Eisenlohr,<sup>4</sup> a protracted delirium with dizziness, vomiting, and ophthalmoplegia (showing Wernicke's syndrome), there were haemorrhagic foci in the region of the third ventricle and of the aqueduct at least one to two centimetres in diameter. Bonhoeffer has described a case of Korsakoff's psychosis, which resulted fatally, in which a large haemorrhagic area was found in the cerebellum.

As a rule the changes are of a noninflammatory nature, but it must be borne in mind that the precise significance of many of the anomalous cells found in these cases has not yet been established. I can only refer you again to Alzheimer's monograph, already referred to, and also to some recent work of Perusini<sup>5</sup> on the *Körnchenzellen* done in Alzheimer's laboratory.

Changes in the neighborhood of the nuclei of other cranial nerves are known, and it is highly probable that many of the cases of sudden death are due to involvement of the vagus nucleus. Thomson, Gudden, and Strümpell have reported such findings.

Changes in the spinal cord are no less striking

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<sup>4</sup>Deutsche medizinische Wochenschrift, 1892. No. 47.

<sup>5</sup>Folia Neurobiologica, i, p. 384.

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than those in the cortex itself, especially degenerations in the columns of Goll and Burdach. Degenerative changes in the intermedullary portions of the anterior horn cells, in the pyramidal tracts, and the lateral cerebellar tracts, have been described by Heilbronner.

Summarized then we see that a diffuse, severe, destructive process is at the foundation of Korsakoff's psychosis; that such process involves the entire nervous system very irregularly, including the parenchyma, bloodvessels, the fibres, with the frequent occurrence of small haemorrhagic foci. Such a process is not pathognomonic for Korsakoff's psychosis by any means; certainly, in the acute stages, the clinical as well as the pathological picture is not distinct from other acute intoxications. However, study may show that the chronic processes are characterized by types of localization not found in other mental disorders, but at best the research involved is time consuming and extremely difficult.

With such a varying pathology, withal so definite, and apt to follow certain trends, the symptomatology becomes all the more interesting to interpret.

Let me call your attention to another case which you saw in the Bellevue wards about three weeks ago, and which you now find in Ward's Island. I shall be very brief with this case.

CASE II.—L. W., forty-two years of age, married, housewife, two children. She had been a moderate drinker for several years, but of late had lost powers of self control and was taken to Bellevue in an attack of delirium tremens, which was not her first offense.

She had been having a steadily advancing neuritis for a short time previous to her admission, and while there as you saw her, you will recall an active delirious patient, with hallucinations of sight and of hearing, the former predominating, and yet unable to move her arms or legs, by reason

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of a flaccid paralysis, and with lost knee jerks. This patient's pupils were unequal, were sluggish to both sight and accommodation, but there was no marked Argyll Robertson pupil.

After her initial delirium she did not clear up as is usually the case—Bonhoffer tells us that about three per cent. of the cases do not, and go on in the manner in which this case has progressed. It is now four weeks since we saw the case in Bellevue while in its delirious phase, which phase is present in from fifty to sixty per cent. of the cases, and in which condition many of the patients die. She still presents the signs of a multiple neuritis, although the paralysis is not so marked as it was, and the indications are that she will make a partial recovery at least.

The chief interests in her case consisted in the mental symptoms. She had been more or less somnolent throughout the time and had shown during the past week unmistakable signs of asymbolia; i. e., she had not known her bed, put her clothes on upside down, and did not recognize which clothes were which.

At the present time she speaks clearly but has been paraphasic and paralexic; even now she reads badly, only partly understanding what she reads. You may recall another patient that I showed you who tried to read with the newspaper reversed. She is badly oriented as to time. Does not know the year, or month, or day; nor can she tell how long she has been here, although she seems perfectly clear superficially. It may be of interest to recall that all of the sensory perceptions may be involved, or, as in a case described by Liepmann, the patient was inattentive to and unable to grasp optic perceptions only.

She has a mild degree of retrograde amnesia, yet you find her comparatively clear for events which antedate her last drinking bout. Her memory for recent events is very bad. She cannot remember anything ten minutes. What she had for lunch has gone from her recollection.

You noted an interesting feature of her confabulations. She was all the time talking about her children. She told you she had been playing with them this morning, and was apparently in a comparatively happy mood notwithstanding her deplorable condition. She gave a number of pseudoreminiscences regarding her husband and children. The type of confabulation here was of the opportunity type, rather than of the suggestive variety which we saw in another case, J. K. You noted she was less liable to take

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hold of my suggestions, but wandered on in a sort of fantastic dream about the children.

For the past week the patient remained apathetic, and had no initiative. As in our other case, it was possible to gain her attention quite easily, and the quick response gave one the idea of an active intelligence, but you noted the attention soon wandered, and in the wards she was never able to go on with anything she started to do. There was a distinct difficulty in her ability to grasp ideas. There was no change in the patient's ability to reason on ordinary topics; her personality was not altered, and beyond the memory defects, and the confabulation, she seemed well mentally. Her mood was for the most part happy, but toward evening she was apt to get anxious, and at times cried very easily.

Permit me now to call your attention to the neuropathological disturbances which may occur in Korsakoff's syndrome. The cases which I have shown you exhibit a number of anomalies to be noted, but you are probably aware—from your work in neurology—how very extensive, and yet how variable may be the changes in multiple neuritis.

*Cranial Nerves.*—So far as changes in the sense of smell are concerned there are no observations on record.

The optic nerve is frequently involved. There may be a temporary or a permanent optic neuritis. Wernicke has recorded retinal changes similar to those found in acute hydrocephalus. Optic neuritis with haemorrhage has been observed, and irregular pupillary pallors are frequently seen. Central scotomata to certain colors, a not infrequent picture of the neuritis retrobulbaris, has also been found.

Inasmuch as there seems to be no doubt that a true optic neuritis may be seen accompanying Korsakoff's syndrome it behooves one to be on one's guard relative to the distinctive diagnosis from tumor, for instance, abscess, or meningitis. The occurrence of a temporal pallor, reported by Bon-

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hoeffer,<sup>6</sup> Oppenheim,<sup>7</sup> Strümpell,<sup>8</sup> and others should be borne in mind in the distinctive diagnosis from multiple sclerosis.

Pupillary disturbances are so common as to have been recognized by almost all observers. Dreschfeld, Kiefer, Gudden, Möli, Thomsen, *et al.*

It is peculiar, however, that little mention of them is made in English literature. White<sup>9</sup> is practically the only American author who notes them. The pupils are frequently unequal. The reaction may be slow; there may be true Argyll Robertson pupil,<sup>10</sup> or there may be paralysis of accommodation. Oppenheim has reported such a case,<sup>11</sup> and I have had one in my service at the City Hospital showing the phenomenon, which cleared up in about four weeks. Myosis is the rule, the pupils being very small. Nonne,<sup>12</sup> however, speaks of a case with marked dilatation of the pupil. I have seen several cases in the Bellevue psychopathic ward, especially in patients suffering from the initial delirium.

Weakness of the eye muscles is common, the abducens seeming to suffer more than the others. Nystagmus is not rare, and ocular palsies not infrequent. In view of these complicating eye symptoms it may be seen at once that the distinctive diagnosis from paresis, or Wernicke's polioencephalitis superior comes into review. In the presence of disturbances of associated eye movements the diagnostic difficulties become even more acute.

Pain in the trigeminus is not rare.

Speech difficulties are very frequent. They are

<sup>6</sup>Die akuten Geisteskrankheiten der Gewohnheitstrinker, 1901 (one of the best of recent monographs).

<sup>7</sup>Berliner klinische Wochenschrift, 1890, No. 24.

<sup>8</sup>Archiv für Psychiatrie, 1883, No. 14.

<sup>9</sup>Outlines of Psychiatry, 1908.

<sup>10</sup>Starr erroneously states that the Argyll Robertson pupil is absent in multiple neuritis, as does also Church. Gowers is less positive.

<sup>11</sup>Zeitschrift für klinische Medizin, xi, p. 234, Case vi.

<sup>12</sup>Jahrbuch der hamburgischen Städtischen Krankenanstalt, 1890.

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complex in causation. The facial, hypoglossal, and subcortical innervation may be severally involved. Bonhoeffer (*l. c.*) states that difficulties in tongue movements are common. This is my own experience. Roth<sup>13</sup> and Kast<sup>14</sup> report lingual atrophy. Such findings are rare.

The facial nerve is not spared. Whereas its implication is not an infrequent accompaniment in multiple neuritis without mental involvement, it seems to be rare in Korsakoff's syndrome. Knapp, in his recent monograph, states that he has never observed it. Paralysis of the soft palate has been seen by Bonhoeffer. Ptosis is not infrequent.

The vagus is frequently implicated; pulse anomalies being exceedingly common. Cyanosis, tachycardia, a characteristic dyspnoea, and short breathing indicate its disturbed action. Frequently the pulse runs up to from 120 to 130, while Knapp (*l. c.*) reports cases of vagus irritation, the pulse dropping to 38 beats to the minute, accompanied by marked reduction in temperature.

Bulbar symptoms, difficulty in swallowing, etc., are rare, but have been observed.

So far as the upper extremities are concerned, they are less frequently involved than the lower. Pain, paresis, or paralysis with atrophy, especially in the distal portions, are to be expected, and there is frequently marked ataxia in the arms.

The lower extremities are usually more severely implicated. The gait is frequently widespread, or halting, occasionally steppage; rarely it shows the cerebellar swaying. Occasionally it is typically tabetic in character. The peronei are most often involved, although Knapp's experience points in other directions. The quadriceps are also implicated,

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<sup>13</sup>*Correspondenzblatt für schweizer Aerzte*, 1902.

<sup>14</sup>*Archiv für klinische Medizin*, xl.

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Bonhoeffer says, as frequently as the peronei. The knee jerks are usually lost, although in a few anomalous cases they are increased, and Knapp reports a case of ankle clonus.

I have already alluded to a case with quadriceps paralysis, the peronei remaining comparatively active.

The sensory and trophic disturbances are in accordance with what is known for alcoholic neuritis.

The bladder is not infrequently involved.

It may thus be seen from these symptoms that a distinction from tabes dorsalis may be necessary. Symptomatically this may be impossible in the early stages. Furthermore, the two conditions may co-exist.

Temperature depression is the rule. This may be due, says Knapp, to an involvement of the vasomotor centres in the medulla. This depression may exist for months, the temperature remaining about 95° F. for all this time. Such subnormal temperatures are noted in cases of mental disturbance associated with brain tumor, and abscess, and in paresis. Other vasomotor disturbances, such as transitory oedemas, transitory erythemas, are of occasional occurrence.

*Focal Symptoms.*

Certain focal symptoms are of particular interest. In discussing the pathological findings I called attention to the changes in the brain stem and subcortical ganglia and to degenerations in the fibre tracts of the cortex. I spoke of the haemorrhagic foci that occasionally were present, and also inferentially called your attention to the fact that oftentimes the degenerations in the fibres themselves were clustered or concentrated, so as to form pseudofocal lesions. In many of the chronic cases

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we have to consider the focal symptoms as largely due to such concentrations of the diffuse degenerations present throughout the brain area. Some of the more important neurological symptoms remain to be discussed.

Speech.—Those of speech are perhaps the most striking. So called stuttering speech of chronic alcoholism, especially in the delirious, is frequent.

- Korsakoff himself, in his original description, which I have already given you, called attention to it. Such speech disturbances may be of purely cortical origin, although Bonhoeffer is inclined to think that they are mostly bulbar.

Motor aphasia.—This has been observed in a number of instances, in fact, many cases of profound Korsakoff's psychosis usually seem to show a motor aphasia. It is of the transcortical type, however, which has been called by various authors a pseudoaphasia (Tiling), a parakinetic aphasia (Knapp), sensory aphasia (Pick), Bischoff, Liepmann, Bonhoeffer, Wernicke, Gudden, have all reported cases in which sensory aphasia of greater or less degree has been observed.

It is natural that in a disorder showing such diffuse changes in the cortex the sensory apparatus should be involved. Knapp has reported a case of word deafness, Bonhoeffer several cases of paraphasia; amnesic aphasia and echolalia are known.

Reading disturbances.—Many cases of Korsakoff's disease showed marked disturbance when reading; false words are read, many small words left out or frequently interjected. Knapp has suggested the term "confabulatory reading disturbance" for the most typical of these changes.

Disturbances in writing are equally present. Perseveration, incoherence, verbigeratory writing are common. Patients very frequently write their

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names over and over again, and the same character of confabulatory feature may be present in the writing as in the speech or reading. Bonhoeffer has reported a case of sensory aphasia agraphia.

Apraxia.—Liepmann's ideatory apraxia is not infrequent in this disorder; such cases have been recorded by Liepmann, Knapp, Gudden, Wernicke, and Bonhoeffer.

Transitory homonymous hemianopsia, word blindness, soul blindness, cortical taste paralysis, asymbolia, agnosia are among some of the rarer disturbances observed.

In a number of cases epileptiform attacks, which may be unilateral or bilateral, occur. These are very frequently associated with an active delirium, are accompanied by unconsciousness, and in most respects resemble an epileptic convulsion. Apoplectiform attacks occur less often, although pseudo-apoplectiform attacks are by no means rare.

*Clinical Forms.*

I have said enough to indicate that great variability exists in the general clinical form in these patients. Truly speaking, the erection of hard and fast types is more or less pernicious in such a kaleidoscopic affection, yet certain trends or tendencies of development are worth recording. This I shall do, telling you the forms described by two authors who have made special studies. Dupré, who in Paris sees all of the prisoners of that large city suspected of any mental disturbance, has written, in the textbook already cited, an excellent résumé of the disease. He gives five clinical forms: (1) *Amnesic form*, in which the profound involvement of the memory stands in the foreground. (2) *Confusional form*, in which the mental haziness remains, the patient remains heavy and stupid, speaks

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slowly, vaguely, and hesitatingly; is inert, indifferent, unstable, undecisive, and anxious. (3) *Delusional form*. Here the patient shows a characteristic dreamy delirium. The combination of loss of memory, mental confusion, suggestive hallucinations, loss of critical power, automatic pseudosensory dreaminess—makes the patient carry on a line of capricious and incoherent waking life. The patients are frequently attacked with delusions of persecution; or have ideas of negation, or of a depressed hypochondriacal type. These are usually fleeting conditions, but may form the basis of a secondary systematized delusional state. (4) *Anxious form*, characterized by the predominance of ideas of disquietude, of anxiety, of panphobias, and active emotional reactions. Emotional paroxysms usually are worse at night. The depressions are largely affective rather than intellectual. (5) *Demented form*, in which the major trend is toward marked intellectual enfeeblement. The asthenic and typhoid types of this form usually lead to a more or less rapid death.

Knapp's monograph, already cited, distinguishes: (1) *Delirious form*, which is present in the beginning at least of two thirds of all patients with this psychosis. (2) *Stuporous form*, quite comparable with Dupré's Confusional Form. (3) *Demented form*. (4) *Hallucinatory form* with hallucinatory excitement and hallucinatory confusion, without any systematization of false ideas. (5) *Hallucinatory form* with systematization, quite comparable with Wernicke's acute hallucinosis. Ideas of reference and anxiety producing hallucinations are frequent. Ideas of injury arise, and ideas of persecution are not infrequent. The confabulation in these patients is filled with ideas of injury. Many of these cases are diagnosticated as paranoias, especially if the

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neuritis is comparatively mild. (6) *Paranoid forms* with episodic attacks of delusional interpretations or pseudoreminiscences of being persecuted. Illusions are not infrequent, and the patients are continually explaining their sensory perceptions by the delusional assumption of external ideas of influence. Ideas of reference and of injury are frequent; being electrocuted, magnetized, etc., are not infrequent; being hypnotized by their enemies they are unable to walk, etc. (7) *Anxiety forms*. (8) *Expansive forms*, in which ideas of grandeur appear in the confabulations. These cases are frequently diagnosticated as cases of paresis, and enter into the statistics of some physicians as "cured cases of paresis." (9) *Manic and melancholic forms*. Such are rare. (10) *Polyneuritic motility psychosis* of Wernicke is a type well recognized by those acquainted with Wernicke's teachings. It is a mélange with characteristic motility changes, into which we cannot enter here. (11) *Anomalous forms* with irregular psychical anomalies. Some patients show impulsive ideas; others a peculiar foulness of thought—obscenities constantly obtruding themselves upon them—others show typical *Vorbeireden*.

*Diagnosis.*

Little, in addition to what has already been said, need be taken up in the discussion of the diagnosis. The chief danger point is the elimination of general paresis, and mistakes may occur either in the prodromal period, during the acute initial state, during the chronic stages, in which the patient's disorder remains stationary, or in the terminal phases.

Irregularity and excessive mixture of symptoms with bizarre groupings make one think of paresis rather than of a Korsakoff, but it should not be for-

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gotten that there are a number of cases of true paresis which run a course very similar to that of a Korsakoff, so that not only is it essential that one should not mistake a Korsakoff for a paretic, but the obverse is true, although less liable—one should not falsely diagnose a paretic as a Korsakoff. Naturally the use of spinal puncture and the cytological examination of spinal fluid has cleared up a number of the difficulties with which psychiatrists formerly had to deal, and has given a precision which has heretofore been lacking. The Wassermann-Plaut serum reaction should also be tried in all doubtful cases.

In the acute delirious stages, Korsakoff's psychosis may be mistaken for a number of things, and it is practically impossible to state with certainty just what the condition may be. Thus the distinction of Korsakoff's delirium from a recurrent delirium tremens or a polioencephalitis haemorrhagica superior may be impossible. In the stuporous forms and apoplectiform forms, acute internal hydrocephalus and meningitis may have to be borne in mind.

*Course.*

The course of this disorder is extremely variable. Many patients, following the acute delirium, enter into a chronic, stuporous state, and die in from two to three weeks; others go on in a less stuporous apathetic condition, gradually growing weaker and weaker, and at the end of three or four months die. These patients frequently show the accompanying effects of disturbance of the liver and kidneys. There may be ascites, oedema, albuminuria, etc. Some patients die of quickly developing miliary tuberculosis.

A comparatively large proportion gradually recover from the effects of the acute delirium and go

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into different stages of restitution. The grade of restitution is impossible to tell in advance.

The age of the patient, the number of attacks of delirium tremens, the physical condition, the cause of the neuritis, whether alcohol or one of the many other causes—all these determine in some measure the outcome.

It may be said that but few patients make a complete recovery. A number make such recoveries that only a skilled observer can detect the impairment, but impairment in some degree usually persists. Still, after a number of years of careful treatment, in patients who may be carefully treated, a restitution *ad integrum* may be possible.

The most frequent cures are those in which only a slight intellectual weakness persists. The amnesias clear up, but the patients remain more or less incapable of sustained, efficient effort. They may become good dilettantes in intellectual pursuits, but rarely accomplish much. Every patient is worth working for, however, and when defects are found to exist, psychological pedagogy may be interested to repair the defects by building up associations in heretofore unused tracts which presumably have not suffered any loss.

*Treatment.*

The treatment differs but slightly from that which you already know to be laid down for multiple neuritis. The preliminary indication is to allay irritability as much as possible, further treatment should retard degeneration, conserve function, and finally aim to educate the damaged organ to better working. In order to allay irritation, hot baths are useful, both to control the delirium, and to limit the pain; hot packs may be used if a hot bath is not at hand. Other eliminative measures in addition

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to heat should be encouraged for a certain time. Thus fair catharsis and active diuresis should be encouraged, since there is a marked tendency for serous extravasations, which may be even haemorrhagic, into vital centres, and it is essential to keep the blood pressure within normal limits. This is usually best accomplished by heat, but it may be necessary to keep the internal and external capillaries fairly well dilated by pressure reducing drugs—drugs that cause excessive vomiting are prejudicial.

It is highly important that the nutrition of the patient be maintained. Overfeeding, as soon as the stomach will permit, is a *sine qua non* in the treatment of Korsakoff's psychosis, which overfeeding should include a high percentage of fat. Milk, butter, and eggs are the most palatable of these foods.

Whether there is any direct lesson to be read from the teachings of Meyer and Overton concerning the relationship of fat and alcoholic absorption I do not know, but it seems reasonable that if the absorption of alcohol in the nervous system stands in more or less direct relation to the solubility of the alcohol or other narcotic in fat, that one should bear this word picture in mind, and realize that fat destruction is an integral portion of the pathology of Korsakoff's disease.

Whether other forms of fat producing food would be better than those that I have mentioned I do not know; that is one of the problems of constructive metabolism.

The neuritis needs to be treated by methods with which you are undoubtedly familiar, massage, electricity, and strychnine.

As to the training of the memory, little progress has yet been made so far as the acute stages are concerned. The patient's grasp of things is so

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slight and so evanescent that applied pedagogy will bring its own reward in loss of time and effort; but later on in the course of the affection a true scientific pedagogy may need to be called upon, especially if the defects observed are more or less localized. If it is found, for instance, that the patient's optical memories seem to have borne the brunt of the storm and have been left with reduced capacity it may be desirable to apply the principles of a constructive education to other less affected receptors or their connections. In these cases each patient is naturally a law unto himself, but there is little doubt that many cases of Korsakoff, which seem to be helpless, and which go on to a terminal dementia for lack of initiative on the part of the physician to search out what part of the mechanism may still be utilizable, might be helped if the principles of scientific pedagogy could be applied.

In the actual treatment of these patients, after the neuritis has recovered to such a degree as to permit of a more or less free going about, occupation cures promise the most. For the more well to do, outdoor games, horseback riding, golf, swimming, are desirable. Carpentry, blacksmithing, bookbinding, or even manual labor on a farm, or training in the open, is advisable.

One principle should not be forgotten in all your therapeutic efforts, and that is not to overdo them, bearing in mind that excessive physical work calls for an amount of nervous functioning just as mental work, that no adaptation to hit even a golf ball correctly can be carried out without an immense amount of nervous functioning, and that therefore in the earlier stages those games or occupations should be chosen which do not call for a high degree of nervous tension—such as the lazy lolling about of swimming—and that no exercise treatment

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should be overdone. Hard muscles, ruddy cheeks, ability to run twenty-five miles after a dog cart, these may be the delusive signs of a healthy musculature at the expense of mental restitution.

Naturally I need not insist upon the avoidance of all tonics which contain alcohol in any form, especially such tonics as peruna and the ilk, some of which I have known to be responsible for the development of Korsakoff's psychosis.

64 WEST FIFTY-SIXTH STREET, NEW YORK.